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14. ABSTRACT: In previous studies, repeated 5-s exposures of anesthetized swine to an electronic control device (TASER International's Advanced TASER® X26 device) resulted in acidosis and increases in blood electrolytes. In the current study, experiments were performed to investigate effects of longer continuous exposures to a different electronic- control-device waveform. After intramuscular injection of tiletamine HCl and zolazepam HCl, anesthesia was maintained with propofol infusion. Ten swine were exposed to either 30- or 60-s applications of an electronic waveform similar to the TASER-X26 device. Transient increases in hematocrit, potassium and sodium were consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise. Blood pH was significantly decreased after exposure, but subsequently returned to baseline levels. Lactate was highly elevated and remained somewhat increased even after three hr post-exposure. Serum myoglobin was increased after exposure and remained elevated for the 3-hr follow-up period. The acidosis would appear to be one of the major concerns regarding long-duration (e.g., several min) exposures in a short period of time. Even with the extremely low pH immediately after exposure, all animals survived. On the basis of the results, further development of useful continuous-exposure electronic control devices is at least feasible, with the caveat that some medical monitoring of subjects may be required.

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conducted electrical weapon, electronic control device, TASER, electro-muscular disruption, muscle contraction

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ORIGINAL ARTICLE

Acute effects of an alternative electronic-control-device waveform in swine

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Abstract In previous studies, repeated 5-s exposures of anesthetized pigs to an electronic control device (TASER International's Advanced TASER® X26 device) resulted in acidosis and increases in blood electrolytes. In the current study, experiments were performed to investigate the effects of longer continuous exposures to a different electronic-control-device waveform. After intramuscular injection of tiletamine HCl and zolazepam HCl, anesthesia was maintained with propofol infusion. Ten pigs were exposed to either 30- or 60-s applications of an electronic waveform similar to the TASER-X26 device. Transient increases in potassium, and sodium were consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise. Blood pH was significantly decreased after exposure, but subsequently returned to baseline levels. Lactate was highly elevated and remained somewhat increased even after three hrs. Serum myoglobin was increased after exposure and remained elevated for the 3-h follow-up period. Acidosis would appear to be one of the major concerns with long-duration (e.g., several min) exposures over a short period of time. Even with the extremely low pH immediately after exposure, all animals survived. On the basis of these results, further development of useful continuous-exposure electronic control devices is at least feasible, with the caveat that some medical monitoring of subjects may be required.

Keywords Forensic · TASER · Electronic control device · Conducted electrical weapon · Electro-muscular disruption · Non-lethal weapon · Acidosis · Myoglobinemia

Introduction

Several reports of TASER® electronic control (alternatively referred to as "electronic incapacitating," "electro-muscular disruption," "electro-muscular incapacitation," "electro-muscular incapacitating," or "conducted electrical weapon") device (ECD) use by law-enforcement personnel have involved repeated applications to an individual over a short period of time. Jauchem et al. [1] published the first study of blood parameter changes after such exposures in an animal model (swine). Blood pH was significantly decreased for 1 h following 18 repeated TASER-X26 ECD exposures, but subsequently returned toward a normal level. ("X26" is a trademark of TASER International, Inc., Scottsdale, AZ. TASER® is a registered trademark of the company.) Lactate was markedly elevated, with a slow return toward baseline. In a subsequent study of only three repeated exposures [2], pH and lactate were significantly changed, but to a lesser degree than in the previous study.

The initial portion of the X26 device waveform (the "arc phase" [3]) is a very high-voltage short-duration pulse designed to penetrate clothing. It serves as a low-impedance electrical conductor that directs a second phase of the waveform of the X26 ECD into the body. The initial phase is not important in generating the maximum degree of muscle contraction in an anesthetized swine model [4].

Although ECDs are often applied a single time for only 5 s, such devices may eventually be deployed regularly in a manner resulting in longer exposures [5–7]. In addition, the

The views and opinions expressed in this article are the authors' own and do not necessarily state or reflect those of the U.S. Government.

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waveform of such devices may change in future models (e.g., some ECDs have included the initial arc phase; others have not). Other investigators have performed initial studies of longer ECD applications [8–12], specifically either two consecutive 40-s exposures or one continuous 80-s exposure.

The present experiments were performed to investigate effects of 30- or 60-s exposures to a new specific waveform (similar to that of the X26 ECD, but without the arc phase) produced by a laboratory electronic stimulator. Muscle contraction and changes in blood parameters were examined in anesthetized swine.

Materials and methods

Animal model

All experiments and animal care procedures were approved by the Institutional Animal Care and Use Committee of the Air Force Research Laboratory, Brooks City-Base, TX, USA, and were conducted according to the U.S. National Institutes of Health's "Guide for the Care and Use of Laboratory Animals," prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources—National Research Council.

Ten male domestic pigs (*Sus scrofa domestica*), ranging in weight from 56.8 to 63.8 kg (mean ± standard error of the mean, 59.3 ± 0.8 kg), were used for these studies. The pig model was selected for several reasons, including similarities to humans in terms of chemical and physical characteristics of blood, respiratory parameters, and responses to muscular exercise [1, 2].

Anesthesia and experimental set-up

For each experiment, a pig was given pre-anesthetic (atropine 0.05–0.5 mg kg^{-1} body weight, subcutaneously) and analgesic (buprenorphine, 0.005–0.02 mg kg^{-1} body weight, intramuscularly) 10–15 min prior to induction of anesthesia. (The buprenorphine was administered to facilitate placement of an endotracheal tube.)

The animals were anesthetized with an intramuscular injection of tiletamine HCl and zolazepam HCl (Telazol®) (6 mg kg^{-1}), followed by oral endotracheal intubation, with the tube secured to the maxilla or mandible. An aural intravenous catheter (3/4–1 in 20–22 ga.) was placed and secured with a cyanoacrylate adhesive and tape. Anesthesia was maintained with 100–125 μg kg^{-1} min^{-1} (or to anesthetic effect) of propofol (PropoFlo®, Abbott Laboratories, North Chicago, IL, USA) delivered by a Baxter syringe pump.

Aspects of propofol relevant to these experiments, compared with other anesthetics, have been discussed previously [1, 2]. Although propofol is often used in humans for moderate or deep sedation rather than full anesthesia [13], the Telazol-induced, propofol-maintained anesthesia used in the present experiments was considered to be appropriate for animals exposed to ECD output. Although deep sedation of humans with propofol can result in some depression of respiration [14], the incidence of apnea has been lower in animals receiving propofol than in those administered other agents such as ketamine [15]. Infusion of propofol in swine has resulted in apnea, but only at a dose rate higher [16] than in our experiments.

Depth of anesthesia was verified by nasal septum pinch, coronary band hoof pressure, and jaw tone. Absence of both reflexes and lack of jaw tone were taken to indicate that the animal was at a suitable level of anesthesia. A jugular venous catheter was placed for subsequent blood sampling. Each pig was delivered to the laboratory anesthetized, placed on its dorsal surface in a canvas sling. At the conclusion of the day's experiment, each animal was euthanized with pentobarbital sodium (Nembutal®), 100 mg kg^{-1} intravenously, without regaining consciousness.

The force of muscle contraction was measured and recorded with a structure (modified from one used by RA Stratbucker, personal communication, 2004), which included a framework constructed of a Unistrut® metal framing system (Unistrut Construction, Wayne, MI, USA). A sling (to contain the pig), pulleys, strain gauges (Model SSM-HA-150, Interface Inc., Scottsdale, AZ, USA), and 3/8″ × 16″ zinc eye-to-eye turnbuckles (Crown Bolts Co., Cerritos, CA, USA) were mounted on the system. Each anesthetized pig was placed on its dorsal surface in the sling. Twisted polypropylene truck rope (3/8 in. diameter, Model 87054, Wellington, Madison, GA, USA) was attached to each limb via a neoprene tennis elbow support (Wal-Mart Stores, Inc., Bentonville, AR, USA), while the other end of the rope was attached to a turnbuckle and strain gauge. A second set of ropes was attached to each limb with neoprene-blend adjustable wrist/elbow supports (Model 483746, BD Consumer Healthcare, Franklin Lakes, NJ, USA). Each of these latter ropes ran through a 4 in. diameter sheave block (Tuf-Tug Products & Accessories, Model SB3000FM, Moraine, OH, USA) and were attached to a 2.27-kg (5 lbs) mass. The output of the strain gauges was quantified, displayed, and stored using equipment and software made by DATAQ Instruments, Inc. (Model DI-720-USB data acquisition system and Version 2.67 WinDaq/Pro + software, Akron, OH, USA). Prior to each exposure, the turnbuckles were adjusted to bring the pig's limbs to a standardized anatomical position (stretched maximally), with a baseline force of approximately 44.5 N (10 lbs).

Heart rate, respiratory rate, and pulse-oximeter oxygen saturation (SpO_2) were monitored continuously using a pulse oximeter (VetOx® G2 Digital, Heska Corporation, Fort Collins, CO, USA), with the probe placed on an ear. Electrocardiogram (ECG) monitoring electrodes (Model 9300-032-50, Mortara Instrument, Milwaukee, WI, USA) were attached the left and right forelimb and to the left hind limb. A lead II ECG signal was amplified by a CWE, Inc., AC/DC bio-amplifier (Model BMA-931, Ardmore, PA, USA) and displayed and stored by the DATAQ Instruments, Inc., hardware and software. In each trial, a baseline ECG was observed before the ECG electrodes were disconnected from the DATAQ hardware to prevent potential damage to the system. (Because of electrical interference, ECG tracings were not readable during ECD discharges.) After ECD discharge, the ECG electrodes were reconnected to the DATAQ recording system, to determine whether asystole or ventricular fibrillation occurred.

Blood sampling and analysis

Jugular venous blood samples were drawn (3 cc each) within 1 min before and 1 min after each ECD exposure, and at other time points, for measurement of pCO_2 and pH. In addition, oxygen partial pressure (pO_2), lactate, glucose, hematocrit, sodium, potassium, and calcium were measured. An additional 9 cc of blood was drawn and allowed to clot at room temperature for at least 30 min. Within 90 min of collection, the samples were centrifuged, and serum was refrigerated until assay.

Levels of whole blood parameters were measured with a GEM® Premier™ 3000 blood gas/electrolyte analyzer (Instrumentation Laboratory, Inc., Lexington, MA, USA). Serum cardiac troponins, creatine phosphokinase (CPK), and lactate dehydrogenase (LDH) (including isoenzyme forms) were evaluated by AniLytics® Incorporated (Gaithersburg, MD, USA). Electrochemiluminescent immunoassays and an ORIGEN® analyzer (IGEN International, Inc., Gaithersburg, MD, USA) were used to analyze cardiac troponins T and I. Streptavidin-coated beads, incubated with a mixture of sample, biotinylated anti-troponin, and ruthenium labeled anti-troponin antibodies, form the basis of these sensitive assays, suitable for detection of the troponins from different animal species.

ECD exposures

The skin was pierced with standard TASER darts (TASER International, Scottsdale, AZ, USA). One dart was placed approximately 5 cm to the right of the midline (approximately 13 cm cranially from the xiphoid process); the other was approximately 7 cm left of the umbilicus (resulting in approximately 30 cm separation between darts diagonally). The darts were connected to the electronic stimulator via X26-device wires.

A modifiable electronic stimulator was previously developed to allow simulation of a wide variety of potential electronic control waveforms. An illustration of the waveform used in the present experiments was presented previously [4, 17]. The stimulator is a small low- to moderate-energy repetitively pulsed electric pulse generator that will allow pulse amplitude, pulse duration, and pulse repetition rate to be varied. The device may be configured to deliver either a single pulse or a series of pulses at an adjustable repetition rate. A thyratron is used to precisely control the repetition rate, which, in the present experiments, was set at 19 Hz. The intent of the design was not to replicate exactly pulses of the X26 ECD, but rather to approximately match the first positive and negative swings in current. Differences of the modifiable device (compared with the X26 ECD) were: (a) a quicker extinguishing of the initial sinusoidal ring, (b) the RC (resistor/capacitor) discharge amplitude not reaching as high a level, and (c) a slightly higher and longer tail to the discharge.

Current output from the ECD was measured through a 1000-Ω resistive load with a Pearson Electronics (Palo Alto, CA, USA) Current Loop Detector (Model 110). The current was recorded on a Tektronix, Inc. (Beaverton, OR, USA) TDS-3052B Oscilloscope. The data were stored on floppy disks for later analysis. Voltage at the output from the stimulator power supply was measured through a voltage divider with a Tektronix TDS-2002 Oscilloscope, to obtain the pulse repetition rate.

To verify that animals would respond adequately to ECD exposure in terms of muscle contraction (i.e., consistent with previous studies [1, 2]), an initial 5-s exposure to an X26 ECD was performed for each animal, followed by a 30-min recovery period. (Rather than allowing full recovery of all blood parameters to baseline, which would have altered the time course for each animal, the experimental protocol was designed with an equivalent standardized recovery time after X26-device exposure.) Pigs were then exposed to the output of the modifiable electronic stimulator (simulating the waveform of the X26 ECD, but omitting the "arc phase") for either 30 or 60 s. Ten animals were exposed in this manner (five for each exposure time), with blood samples taken for 3 h afterward. Exposure times were alternated on each day of the experiment.

Statistical analyses

Due to the small N sizes, data from the 30- and 60-s exposure groups were not analyzed separately, and comparisons were not performed between the two groups. Data from the two groups, however, are presented separately in graphic form (in the "Results" section below).

Data for all ten animals (groups combined) were analyzed by one-way repeated-measures analyses of variance (ANOVAs), in which measurement time was the repeated factor. Post hoc comparisons were performed using the Dunnett approach, with the time point before the initial 5-s exposure as the control. In all analyses, the rejection level for statistical significance was set at $\alpha = .05$.

Results

Muscle contraction force

The initial 5-s exposure to an X26 ECD resulted in a contraction force (calculated by averaging maximal values during the first second of exposure across all four limbs) of 231 ± 10 N. Maximal limb contraction occurred at the start of each 30- or 60-s exposure, with a slight drop in the force during the remainder of the exposure. For the five subjects receiving 30 s of exposure, the mean level of maximal limb contraction was 259 ± 11 N; the mean level of maximal limb contraction for the five animals receiving 1 min of exposure was 311 ± 61 N (large variance due to one outlier).

Whole blood sample changes

In each figure of this paper, sampling time is shown in minutes, with "Pre" referring to before initial X26 exposure and "Imm." referring to immediately after each respective exposure.

Levels of blood lactate and glucose during the experiments are shown in Fig. 1. Lactate was significantly elevated at all post-exposure time points. Glucose was not elevated immediately after the single 5-s X26 exposure, but was significantly elevated at all subsequent points.

In Fig. 2, pH and hematocrit are illustrated. A number of 60-s exposures resulted in pH values less than the lower limit of measurement of the blood-gas/electrolyte analyzer (6.8). Measurements of less than 6.8 were assigned a value of 6.8 for purposes of data analysis. The pH was significantly decreased below baseline values immediately after the 30- or 60-s ECD exposures, but gradually returned toward pre-exposure values. At time points after 60-min post-exposure, there were no significant differences from the baseline value.

Distinctly lower values for hematocrit are common in pigs (compared with humans). The pre-exposure samples in the current experiments were within normal ranges as reported by Hannon et al. [18] in studies of conscious pigs. There were significant increases in hematocrit at all time points after the 30- or 60-s ECD exposures.

Although the venous blood pCO_2 was significantly elevated immediately after exposure (above the measurement range of the blood-gas analyzer of 115 mmHg), it returned

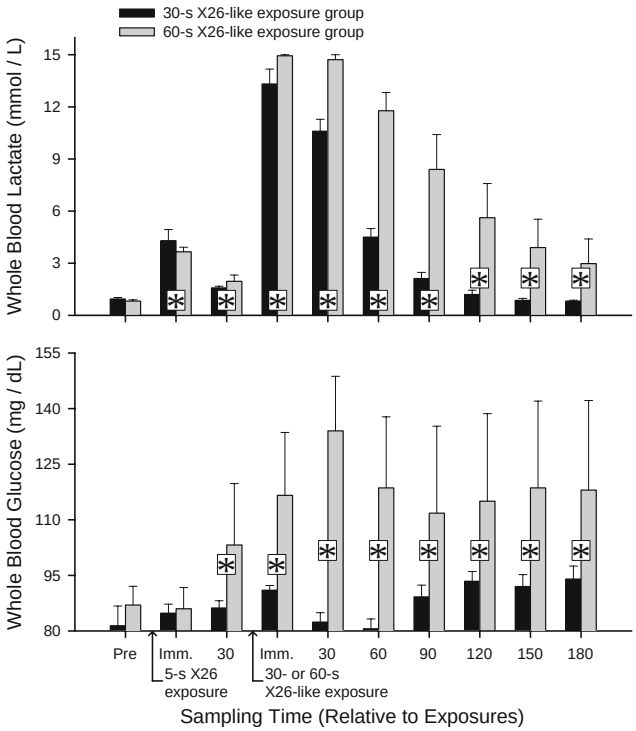

Fig. 1 Jugular venous blood lactate and glucose (mean ± SEM) of swine before and after exposure for either 30 or 60 s (N = 5 for each) to a waveform similar to the TASER X26 device. "Imm." = immediately after exposure. Sampling time is in minutes. A single 5-s exposure to a TASER X26 was performed initially. Due to the small N sizes, data from the 30- and 60-s exposure groups were not analyzed separately, and comparisons were not performed between the two groups. * = total from the two groups combined (total N = 10) significantly different from pre-exposure value

to baseline levels at the subsequent time point. Venous blood pO_2 exhibited no significant changes at any point (pre-exposure baseline level was 56 ± 4 mmHg).

Sodium and potassium levels were significantly increased immediately after exposure, but returned to baseline levels at the subsequent time point (pre- and immediate post-values for both groups combined: sodium, 134 ± 1 and 144 ± 2 mmol l^{-1}; potassium, 4.2 ± 0.3 and 6.2 ± 1.2 mmol l^{-1}). There were no significant changes in blood calcium.

Serum sample changes

Serum myoglobin, as shown in Fig. 3, increased significantly immediately after 30- or 60-s ECD exposures, and remained so throughout the study.

Significant increases in total CPK and in the CPK-MM fraction were seen immediately after 30- or 60-s ECD exposures and at subsequent time points (Fig. 4).

No major changes were found in lactate dehydrogenase isoenzyme levels. Serum levels of cardiac troponin T

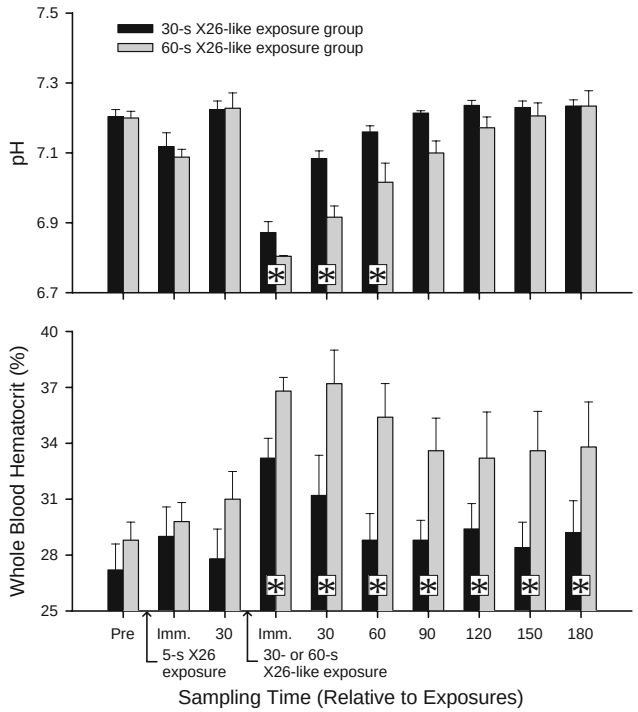

Fig. 2 Jugular venous blood pH and hematocrit (mean ± SEM) of swine before and after exposure for either 30 or 60 s (N = 5 for each) to a waveform similar to the TASER X26 device. "Imm." = immediately after exposure. Sampling time is in minutes. A single 5-s exposure to a TASER X26 was performed initially. Due to the small N sizes, data from the 30- and 60-s exposure groups were not analyzed separately, and comparisons were not performed between the two groups. * = total from the two groups combined (total N = 10) significantly different from pre-exposure value

remained undetectable in all animals. Cardiac troponin I was detected in only one animal (after ECD exposure for 30 s). The level was 0.64 ng ml^{-1} pre-exposure; the highest level was 1.81 ng ml^{-1}, 2 h post-exposure.

Heart rate, respiratory rate, and arterial oxygen saturation

Neither asystole or nor ventricular fibrillation were observed on the electrocardiogram. Heart rate increased following 30- or 60-s exposures, but not significantly (pre-exposure heart rate, before 30- and 60-s exposures, was 97 ± 3 for both groups combined). Although each animal appeared to attempt breathing activity (i.e., slight movements of the chest), no substantial breaths were noted during exposures. Instead, a sustained single inspiration was seen during each exposure. Mean respiration rate decreased immediately post-exposure in each group (though not significantly), and subsequently returned to baseline levels (pre-exposure values were 33 ± 7 and 36 ± 8 breaths/min in 30- and 60-s exposure groups, respectively).

Arterial oxygen saturation exhibited no significant changes during or after the exposures, but increased slightly

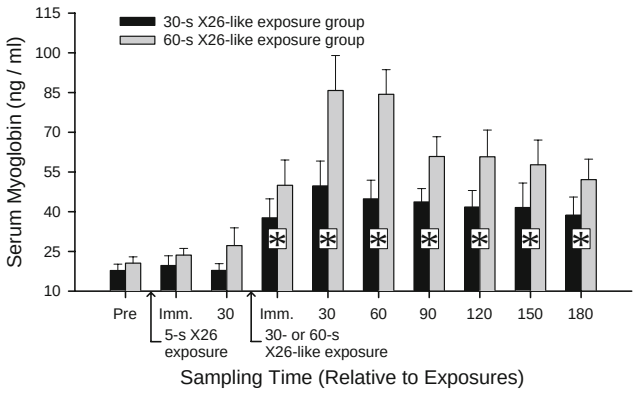

Fig. 3 Serum myoglobin of swine before and after exposure for either 30 or 60 s (N = 5 for each) to a waveform similar to the TASER X26 device. "Imm." = immediately after exposure. Sampling time is in minutes. A single 5-s exposure to a TASER X26 was performed initially. Due to the small N sizes, data from the 30- and 60-s exposure groups were not analyzed separately, and comparisons were not performed between the two groups. * = total from the two groups combined (total N = 10) significantly different from pre-exposure value

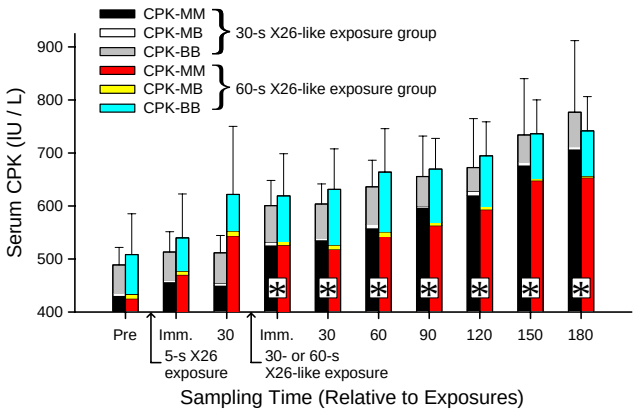

Fig. 4 Serum creatine phosphokinase of swine before and after exposure for either 30 or 60 s (N = 5 for each) to a waveform similar to the TASER X26 device. "Imm." = immediately after exposure. Sampling time is in minutes. Data for the 30-s exposure group are shown on the first bar at each time point; data for the 60-s exposure group are shown on the second bar at each time point. A single 5-s exposure to a TASER X26 was performed initially. This is a "stacked graph," a bar chart with the relative percentage of each isoenzyme represented by a vertical band height (rather than with heights "superimposed"). The total height of each whole bar at a given time point represents the total CPK (i.e., the top of the bars correspond with values on the *y*-axis). Each different grayscale or color portion graphically represents the percentage of each individual isoenzyme (i.e., individual band heights do NOT correspond with values on the *y*-axis). Due to the small N sizes, data from the 30- and 60-s exposure groups were not analyzed separately, and comparisons were not performed between the two groups. * = total CPK from the two groups combined (total N = 10) significantly different from pre-exposure value

in each group in the latter parts of the experiments (from baselines of 92 ± 2 and 90 ± 3% in the 30- and 60-s groups, respectively, up to 97 ± 3 and 96 ± 2%).

Discussion

Muscle contraction

In terms of muscular contraction effectiveness (amount of force generated), the current experimental results during exposures were similar to previous results of exposure to the X26 ECD [1, 2]. The maximum degrees of muscle contraction generated during 30 vs. 60 s of ECD exposure were similar.

Lactate and pH

Blood pH exhibited a significant decrease in each group after 30- or 60-s ECD exposure. After approximately 2 h, however, pH had returned to baseline levels. Although the pre-exposure values of pH were low (approximately 7.2) compared to the previous studies [1, 2], other investigators have also reported control values in pigs as low as 7.2 [19, 20]. (Mixed venous pH [which was measured in our studies] is usually only about .04 units less than arterial pH [21].) Hamilton et al. [22] reported a pH of 7.18 in pigs after "high-intensity handling." Although Haydon and West [20] found that certain specific diets can lower blood pH in pigs for 5 h (the total time of monitoring in their study), the animals in our present study were fasted for at least 18 h. Any reasons for the disparity between our different studies are unknown. There were no apparent variations in handling or diet between the current and our previous [1, 2] experiments. The specific blood gas analyzer used in these experiments exhibits a high degree of linearity for pH within the reportable range of 6.80 to 7.80 [23].

In the present experiments, blood lactate was highly elevated after 30- or 60-s ECD exposure. In the 30-s exposure group, it had returned to baseline levels after 2 h. In the 60-s exposure group, however, it remained somewhat elevated even after 3 h. Lactate accumulation most likely results from the interaction of many different biochemical and physiological processes rather than purely a deficiency of oxygen (reviewed by Gladden [24]). (See Jauchem et al. [1, 2] for previous discussion of blood lactate changes after ECD applications.)

Some of the factors referenced in the following sections are, in fact, affected by lactate levels and pH, but are mentioned separately for discussion purposes.

Respiratory rate, CO_2, O_2, and heart rate

Pre-exposure respiratory rate and venous blood pO_2 were above the range reported by Hannon et al. [18] in conscious pigs. Cessation of effective breathing, which occurred during ECD exposure, was probably related to direct effects on muscles involved in respiration.

The animals in our study did not exhibit effective respiration during the ECD exposure periods. Other investigators also noted impaired ventilation in anesthetized pigs exposed to the X26 device [8] and to a different ECD [25], thus reporting results similar to ours. Although decreased respiration can be produced in animals under these experimental conditions of ECD exposure, however, the validity of translating these results to human subjects is unknown, as mentioned previously by Jauchem [26]. Human subjects could be aware of experimental protocols (and potential effects of electronic control devices) and could, therefore, be able to consciously focus on and maintain respiration. One investigator [27] questioned whether human subjects were actively instructed to focus on breathing during ECD exposures.

Since venous pCO_2 is similar to arterial values (except in frank cardiovascular shock [21]), the high blood pCO_2 values in the current experiments would be likely to reflect respiratory acidosis. The hypercapnia in the present experiments was short-lived, and pCO_2 was back to baseline levels 30 min after the 30- and 60-s exposures.

Pre-exposure heart rate was within ranges reported by Hannon et al. [18] and Hastings et al. [28] for restrained and unrestrained conscious pigs, respectively. Although heart rate increased after the 30- or 60-s ECD exposure periods, the increases were much less than that seen in (a) other studies of steady-state and exhaustive exercise [28] and (b) the previous study of 18 repeated X26 ECD exposures [1].

One limitation of our present study was the inability to observe activity of the heart during ECD applications. The work of Dennis et al. [8], Valentino et al. [9, 29], and Walter et al. [12], however, has been critical in defining ventricular capture and potentially fatal ventricular arrhythmias that may occur during applications of ECDs. In the first study ever accomplished with echocardiography during ECD applications, TASER X26 ECD exposures had major effects, including capture of cardiac rhythm, ventricular tachycardia, or flutter, and a case of fatal ventricular fibrillation [12]. Upon examination of supplemental material associated with the report of that study (video clip at: http://www3.interscience.wiley.com/cgibin/fulltext/119413611/sm001.avi?PLACEBO=I.E.pdf), a reader may clearly observe some of the echocardiographic results. In another study, ventricular rhythm was captured immediately in a majority of experiments with the ECD applied in transcardiac vectors [29]. Ho et al. [30], who noted no such capture in human subjects, suggested that swine models "may have limitations when evaluating ECD technology."

Hematocrit, electrolytes, and glucose

The increase in hematocrit after ECD exposure was consistent with previous studies of muscle stimulation [31]. The

increases in blood sodium and potassium after exposures, although statistically significant, returned to approximate pre-exposure levels within 30 min. It is doubtful that these short-term elevations would have any serious health consequences in a normal individual.

Bozeman [32] suggested that lethality due to ECD impact could be due to hyperkalemia related to muscle contraction. Exercise-induced and normally fatal levels of blood potassium concentration have been reviewed previously [26]. On the basis of the current study and our previous investigations [1, 2], there may be a wide margin of safety, relative to hyperkalemia, for most ECD applications.

The increase in blood glucose, in animals exposed for 60 s, was similar to that in the earlier series [1] of repeated 18 X26 ECD exposures.

Myoglobin and CPK

In the current study, serum myoglobin was increased after exposure and remained elevated for the 3-h follow-up period. A comparison of myoglobin levels after X26 ECD exposures, with levels after toxic, ischemic or traumatic rhabdomyolysis, has been presented previously by Jauchem et al. [2].

The increases in CPK and in percentage of CPK-MM in the current experiments were consistent with previous findings of similar changes after equine exercise [33] and in pigs following electrical stimulation (and subsequent rapid running for 5 min) [34]. Such increases were also found in swine exposed to only three 5-s applications of X26 ECDs [2]. Sloane and Vilke [35] noted that increases in CPK would not be surprising after ECD application, since massive contraction of muscle could cause such changes.

Overall relevance of results to possible ECD repeated-exposure scenarios

Changes in most blood parameters, including pH and potassium, were more severe than in previous experiments [1, 2] of 5-s applications of ECDs. Acidosis would appear to be one of the major concerns regarding long-duration (e.g., several min) ECD exposures over a short period of time. Although treatment with sodium bicarbonate may counter acidosis, infusion of large amounts may incur some risks [36]; therefore such therapy should only be instituted with great care. On the basis of this study of relatively long-duration exposures to an ECD on blood parameters, it is apparent that, although major changes in lactate and pH occur, animals are still able to survive such scenarios.

Not all animals will survive more "extreme" exposures to ECDs. For example, after 3 min of cycling exposures (7 s on, followed by 3 s off, repeatedly) to the output of the TASER-X26 device (modified for the longer exposure periods), only four out of ten animals survived [37]. Thus, for total exposure times longer than the 60 s of the current study, further development of useful ECDs (for long-term incapacitation during military operations) may require consideration of longer pauses between repeated exposures. As the number of combinations of possible waveform designs and pauses between applications would be infinite, testing of only a limited number of such permutations would be practical.

Conclusion

Transient increases in potassium, and sodium after ECD applications were consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise. These increases, although statistically significant, returned to pre-exposure levels within 30 min. It is doubtful that these short-term levels of elevation would have any serious health consequences in a normal individual. Blood pH was significantly decreased following exposure, but subsequently returned toward a baseline level. Lactate was highly elevated, with a slow return toward baseline. The changes in these factors were more severe than those after previous applications of only three 5-s ECD exposures. Oxygen saturation (measured by pulse oximetry) was not significantly decreased after exposure.

In conclusion, although 30- or 60-s exposures to a specific ECD waveform resulted in significant changes in blood chemistry, levels of most of these parameters returned to baseline. Even with an extremely low pH immediately after exposure, all animals survived.

Educational message

1. Repeated or long-duration applications of electronic control devices can result in physiological changes, including acidosis and increases in blood electrolytes.
2. Different electronic-control-device waveforms may be used in the future.
3. Transient increases in hematocrit, blood potassium, and sodium due to electronic control devices are consistent with previous reports in the literature dealing with studies of muscle stimulation or exercise.
4. Acidosis may be of major concern regarding long-duration (e.g., several min) exposures to electronic control devices in a short period of time.
5. Further development of useful continuous-exposure electronic control devices is at least feasible, with the caveat that some medical monitoring of subjects may be required.

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